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Original Research Article

Value of Cardiac Magnetic Resonance Imaging in Evaluation of Cardiac Function and Adverse Cardiac Events in Repaired Tetralogy of Fallot Patients

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ABSTRACT

Background: Because of its advantages over cardiac computed tomography (CT) and echocardiography, cardiovascular magnetic resonance (CMR) has developed as an imaging technique for evaluating the functional outcomes of cases who have undergone repaired tetralogy of Fallot (TOF). This study aimed to assess the role of CMR in evaluating repaired tetralogy of Fallot cases.

Methods: In this observational study, we included 33 patients ranging from 2 to 51 years who had repaired the Tetralogy of Fallot using a Philips 1.5 tesla MRI machine and were referred to the diagnostic radiology department at Zagazig University Hospitals. All cases underwent complete history taking, examination, 12 lead ECG, Echocardiography, and CMR.

Results: Right ventricle Ejection fraction ranged from 22 to 66% with a mean of 50.88%. Fifteen patients (45.5%) had good systolic function; eleven patients (33.3%) had impaired systolic function, while seven patients (21.2%) had fair systolic function. Concerning pulmonary valve regurgitation, seventeen patients (51.5%) had a regurgitation factor>40%, which denoted severe regurgitation, and one-third had moderate regurgitation. As regards late gadolinium enhancement, six patients (18.2%) had no enhancement, 69.7% had RVOT enhancement, 54.5% had VSD patch region enhancement, 18.2% had RV insertion area enhancement, 15.2% had enhancement of the RV septal surface except for the VSD patch region. In contrast, 3% had enhancement in the inferior wall of the RV.

Conclusions: Evaluation of cases with repaired Tetralogy of Fallot is greatly aided by cardiac magnetic resonance imaging (MRI). It aids in assessing surgical repair effectiveness, identifying diagnosed complications of residual abnormalities, and directing subsequent operations by giving thorough anatomical and functional information.

Key Words: Cardiac Function, Cardiac Magnetic Resonance, Repaired Tetralogy of Fallot

INTRODUCTION

Four to five out of every 100,000 newborns are diagnosed with tetralogy of Fallot (TOF), making up about 7 to 10 percent of all congenital cardiac abnormalities. A tetrad of anomalies, including ventricular septal defect (VSD), pulmonary stenosis, right ventricular hypertrophy, and aorta overriding

the ventricular septum, are commonly associated with tetralogy of Fallot. However, it has been hypothesized that this is due to a misalignment of the infundibular septum with the muscular septum in the anterior position [1].

The survival rate for cases with TOF has increased to greater than 90% after early childhood, when most patients have complete repair. Failure to thrive and erythrocytosis are common side effects for children who have received palliative operations. Untreated TOF patients have a 50% chance of surviving between 5 and 10 years due to complications such as hypoxemia, endocarditis, brain abscesses, and cerebral vascular accidents. Rarely do people with unrepaired or palliated TOF live past their 40s [2].

Survival rates for patients with repaired tetralogy of Fallot (rTOF) have increased dramatically after surgery. Right ventricular (RV) dilatation, RV dysfunction, ventricular arrhythmia, symptomatic heart failure, as well as sudden cardiac death have all been linked to PR after this treatment. The risk of arrhythmias decreases, RV volume decreases or is normalized and functional class is improved after pulmonary valve replacement (PVR) [3].

When RV function is already compromised, however, PVR does not help. That's why getting the operation done before RV dysfunction becomes noticeable is crucial. Individuals may have a different RV dilatation threshold associated with permanent RV impairment. In addition, there is inconsistent evidence regarding how PVR affects RV remodeling, the duration of QRS, and the risk of arrhythmia [4].

Conversely, one must consider the surgical risks associated with (PVR) and, notably, the restricted lifespan of prosthetic valves, especially in younger patients. Currently, the question remains unanswered regarding whether the advantages of PVR outweigh the complications linked to the procedure and the limited durability of presently utilized prosthetic valves. Furthermore, specific criteria for PVR in the pediatric population (those under 18 years old) or based on gender have not yet been established. [5].

The radiologist plays a crucial role in the healthcare team by interpreting various imaging modalities; this requires thorough familiarity with TOF anatomy, surgical techniques, and probable complications following surgery [6].

Pre and postoperative CHD involving TOF evaluations rely heavily on Transthoracic echocardiography (TTE) from fetal diagnosis to late complications assessment due to the low cost, easy availability, and absence of contraindications. TTE allows for cardiac structure measurement, giving essential information about pulmonary stenosis severity, ventricular septal defect (VSD) size and morphology, aortic override, and RVOT obstruction. It also enables surgical repair assessment, including evaluation of adequate relief of RVOTO, VSD closure, and residual intra-cardiac shunts [7].

However, echocardiography is the first-line modality for the evaluation of rTOF. Cardiovascular magnetic resonance is a gold standard for volumetric assessment and shunt quantification to guide intervention and provide structural assessment challenging to image with echocardiography. In addition, CMR can provide tissue characterization and assess the presence of myocardial fibrosis [7].

Computed tomography (CT) angiography is a noninvasive imaging method that provides a clear picture of the cardiovascular system in minimal time. Also, because it does not use ionizing radiation, magnetic resonance imaging (MRI) is safe for repeated use without worrying about radiation toxicity. It's the ability to create high-quality images of the inside of the heart, with an unlimited number of imaging planes, precise flow quantification, and a readout of how well the ventricles are working [8].

This research was performed to assess the role of CMR in evaluating patients with repaired tetralogy of Fallot.

METHODS

This observational study was conducted at the Radio-diagnosis department for six months from December 2022 to June 2023. The Data were collected from 33 patients with repaired TOF diagnosed clinically and by transthoracic echocardiography (TTE). There were 16 males (48.5%) and 17 females (51.5%). Their ages ranged from 2 to 51 years, with a median age of 7.75. Eighteen patients were (<18 years).

All pediatric patients were under anesthesia, and written informed consent was obtained from their parents. Also, written informed consent was obtained from other adult patients, and the research ethical committee of the Faculty of Medicine, Zagazig University, approved the study. The institution's medical ethics committee approved the study protocol (with reference number ZU-IRB#9755/20-09-2022). The Declaration of Helsinki, issued by the World Medical Association to protect people participating in medical research, was strictly followed during this study.

Inclusion Criteria: We included patients who had primary repair (total surgical repair) of tetralogy of Fallot. rTOF was diagnosed clinically and by transthoracic echocardiography (TTE).

Exclusion Criteria*:* We excluded any patient who had any of the following congenital heart defects: heterotaxy syndrome, common atrioventricular canal, major aorto-pulmonary collateral arteries (because they are small, tortuous vessels that can arise from the aorta and can follow complex paths, making their imaging challenging with standard CMR techniques.), double outlet RV, regurgitation of the aortic and the mitral valve, as well as RV-to-pulmonary artery conduits (due to metallic artifacts as these conduits made of metallic prostheses and also due to some flow-related issues as blood flow within conduits create turbulence which affect quality of imaging.)

All patients are subjected to the following:

Good preparation, training, anesthesia for children cases, entire clinical history taking, age, sex, and any previous cardiac surgery or chronic disease. And about the disease as the age at operation, preoperative cardiac CT, last echocardiography, and renal function tests (GFR should be more than 30 ml/min/1.73 m^2 for patients who underwent the scan with contrast). Clinical evaluation was done for all patients, and ECG leads were connected and placed on cases properly. A peripheral intravenous cannula was done for the cases who had a contrast injection, which is helpful in identifying scar tissue by highlighting regions of myocardial scarring or fibrosis that might impact heart function. Height and weight were assessed to estimate the body surface area (BSA) used in measuring ventricular volumes, and then a dedicated cardiac coil was used.

Imaging protocol:

The protocol was done in 65–70 min. Static (localizing) images of ECG-gated planes (axial, sagittal, and coronal), including Localizers, false two chambers, and false, short axis. Cine (dynamic) images ECG-triggered, free-breathing or breath-hold cine SSFP in four-chambers, RV two-chambers, LV two-chambers, three-chambers, RVOT cross, Oblique sagittal parallel to the RVOT and proximal MPA, Ventricular short-axis and axial plane, LVOT, sagittal RPA, LPA and cine pulmonary sagittal, coronal, as well as Axial stack of the whole heart.

Magnetic resonance angiogram (MRA): Flow images (anatomy and phasing): Measurements of the aortic, main pulmonary artery, proximal right pulmonary artery, and left pulmonary artery (LPA) flow were taken by cine phase-contrast flow (breath-hold). After the contrast dye had been injected and a TI scout had been performed to determine the optimal time of inversion, a late gadolinium enhancement (LGE) sequence was acquired 10 minutes later (Short-axis, phase-sensitive contrast inversion recovery sequences and 2, 3, and 4-chamber in the same planes).

Image interpretation: was conducted asynchronously using DICOM and PACS network connection (local picture archiving and communication system).

Cine images and viewing localizers were used for visual assessment. At the same time, post-processing quantitative analysis included flow analysis, which involved manually drawing the ROI (area of interest) in each image to estimate the flow/cardiac cycle. The results were a flow/time graph through which flow measurements (forward, backward, net flow, velocity, regurgitation fraction, and pressure gradient across the PV and RVOTA) could be obtained using a combination of short axis and axial cine stacks and late gadolinium enhanced pictures; we can calculate ventricular volumes.

T1 and T2 mapping.

Ventriculography: We employed computerized, semi-automated analytic software to examine a stack of short-axis images and axial images. For both ventricles, the end-diastolic and end-systolic phases were selected and completed. During the ED phase, the blood volume in the left or right ventricle was greatest just before the aortic or pulmonary valves opened or the mitral and tricuspid valves closed (respectively). The volume of blood was lowest during the ES phase. Outline the right ventricular (RV) and left ventricle (LV) endocardial borders (including RV trabeculations, LV papillary muscle, and LV outflow pathways) during the ED and ES phases.

Confirmation of results: Phase-contrast imaging was used to assess the severity of pulmonary and aortic valvular regurgitation (by subtracting backward flow at a sub-valvular level from the forward flow at the valve level). RV stroke volume minus forward flow in the pulmonary artery divided by RV stroke volume, an indirect measure of tricuspid regurgitation, was obtained. An indirect measure of mitral regurgitation was obtained by dividing the LV stroke volume by the difference between the aortic forward flow and the LV stroke volume. To calculate pulmonary flow, add the forward RPA flow to the forward LPA flow. In the case of the presence of shunts, Qp/Qs were calculated. RV and LV stroke volumes were very similar since neither was affected in the absence of the shunts or valvular regurgitation. Comparison of late gadolinium enhanced (LGE) images to their corresponding cine images (4 chambers and short axis).

Regarding the grading score of aortic regurgitation: RF <15% and regurgitation volume<30 ml/beat was considered a mild degree, RF 15-29% and regurgitation volume30-59 ml/beat, it was considered a moderate degree and RF ≥ 30% and regurgitation volume ≥60 ml/beat, it was considered a severe degree, regarding the grading score of mitral regurgitation: Regurgitation volume <30 ml/beat and regurgitation fraction <20%, it was considered a mild degree, if regurgitation volume 30-59 ml/beat and regurgitation fraction 20-39%, it was considered a moderate degree, if regurgitation volume ≥60 ml/beat and regurgitation fraction ≥40 %, it was considered a severe degree. As regards pulmonary regurgitation grading score: If RF <20% (mild), RF 20-40% (moderate), and RF >40% (severe). Regarding the grading score of aortic valve stenosis: Mild if valve area> 1.5 cm^3, mean pressure gradient< 25 mmHg, moderate: Valve area 1-1.5 cm^3, mean pressure gradient 25-40 mmHg, and severe: Valve area < 1 cm^3, mean pressure gradient >40 mmHg. Regarding mitral stenosis grading score: Mild: valve area >1.5 cm^3, moderate: valve area 1-1.5 cm^3, and severe: valve area <1 cm^3. Regarding the severity of pulmonary valve stenosis, grading score: Mild: peak gradient <36mmHg, mean gradients <20 mmHg, moderate: peak gradient 36-64 mmHg, mean gradients 20-40 mmHg and severe: peak gradient ≥64 mmHg, mean gradients ≥40 mmHg. Regarding the grading system of the RV function [5].

Normal RV function: RVEF within a normal range, along with normal volumes and absence of significant wall motion abnormalities. Mild RV dysfunction: mild reduction in RVEF, possibly with slight changes in volumes or regional wall motion; moderate RV dysfunction: moderate reduction in RVEF, along with more noticeable changes in volumes and regional

wall motion; and severe RV dysfunction: significant reduction in RVEF, abnormal volumes, and evident wall motion abnormalities [5].

The RV function grading system depends on RVEF, RVEDV, RVESV, and RF in pulmonary valve regurgitation and wall motion abnormalities. According to the grading of pulmonary artery stenosis, a specific grading system may consider several parameters such as Evaluation of blood flow velocity and acceleration, pulmonary artery diameter, pressure gradient, assessment of vessel walls changes and presence of collateral circulation: Peak velocity values above two or 2.5m/s in the main or branch pulmonary arteries suggest significant stenosis, mean velocities higher than 1.5 or 1.7 m/s suggest significant stenosis, and pressure gradients above 30-40 mmHg across the stenotic segment of the pulmonary artery might suggest significant stenosis [5].

STATISTICAL ANALYSIS

The data was analyzed using SPSS (Statistical Package for the Social Sciences) version 26. Categorical variables were defined by their absolute frequencies. The chi-square for the trend test was performed to examine the relationship between the two sets of ordinal data. Parametric test assumptions were checked using the Shapiro-Wilk test. Depending on the data type, quantitative variables were described using mean values, standard deviations, median values, and interquartile ranges. To determine how strongly and in what direction two continuous variables were associated, researchers used the Spearman rank correlation coefficient and the Pearson product-moment correlation coefficient.

RESULTS

There was almost equal gender distribution, and 54.5% were under 18. Regarding presenting complaints among studied patients, a larger percentage had more than one complaint (39.4%) (Table 1).

The ejection fraction of the cases ranged from 22 to 66%, with a mean of 50.88%. Fifteen patients (45.5%) had good systolic function. Mean end-systolic, end-diastolic, and stroke volumes were 115.15 ml, 221.91 ml, and 106.73 ml, respectively. The indexed ESV (iESV), indexed EDV (iEDV), and indexed stroke volume (iSV) were 72.46, 152.48, and 79.3 (mL/m^2), respectively. Patients' left ventricular ejection fractions varied from 30-72%, with a mean of 56.24%. Twenty-two patients (66.7%) had good systolic function. Mean EDV, ESV, and stroke volume were 119.3 ml, 54.56 ml, and 64.28 ml. The mean indexed end-systolic volume, indexed end-diastolic volume, and indexed stroke volume were 35.75, 82.43, and 46.79(mL/m2), respectively. RT EDVI/LVEDVI ranged from 0.86 to 4.17; the mean aorta netflow was 59.11 cm/sec. Median aorta backflow was 1.5 cm/sec, ranging from 0 to 17; eight patients had aorta backflow. The mean aorta forward flow was 60.71 cm/sec. Median aorta regurgitation factor was 0%. All patients with aortic valve regurgitation had mild form (Table 2).

Concerning pulmonary valve regurgitation, seventeen patients (51.5%) had severe regurgitation (Table 3). About 48.5% had mild residual pulmonary stenosis, the mean pressure gradient was 35.31, 33.3% had no septal flattening, and about 69.7% had no residual VSD (Table 4).

Regarding late gadolinium enhancement, 69.7% had RVOT enhancement, and 54.5% had VSD patch region enhancement (Table 5).

A male patient, 16 years old, had a history of total surgically repaired Tetralogy of Fallot, presented with fatigue, and was doing an MRI scan for follow-up and estimation of right ventricular volume and flow. MRI findings: Moderate right ventricular dilatation when compared to LV (RVEDVI: LVEDVI = 2.1:1), RVEDVI= 199 ml/m^2, RVESVI =86ml/m^2, with good systolic function. Evidence of moderate pulmonary valve regurgitation about (34%) with Evidence of origin of LPA severe stenosis (dephasing jet with maximum velocity about 3.5m/s), estimated pressure gradient 49mmHg. Evidence of origin of RPA moderate stenosis

(dephasing jet with maximum velocity about 3m/s), estimated pressure gradient 36mmHg. With evidence of RVOT enhancement, the likely site of the patch and basal inter-ventricular septum is likely the site of VSD closure (Figure 1).

A 14-year-old male patient with total surgically repaired TOF underwent CMR for follow-up. MRI findings: RV is mildly dilated compared to LV (RVEDVI: LVEDVI = 1.9:1), RVEDVI = 214 ml/m2, RVESVI =99ml/m2, with good systolic function (EF = 54%). Evidence of moderate calculated pulmonary valve regurgitation is about (33%) with evidence of moderate LPA regurgitation at 35% and Evidence of right pulmonary artery moderate stenosis (dephasing jet with maximum velocity about 3m/s), with an estimated pressure gradient of 36mmHg. Also, he had evidence of RVOT enhancement, likely patch, and basal inter-ventricular septum, likely the site of VSD closure (Figure 2).

Table (1) Socio-demographic characteristics, presenting complaints and Distribution of associated abnormalities among the studied patients:

	N=33	**%**
Gender		
Male	16	48.5%
Female	17	51.5%
	Median (IQR)	Range
Age (year)	7.75(7 – 11)	2 – 51
<18 years	18	54.5%
≥18 years	15	45.5%
	N=33	%
Presenting complaint		
Asymptomatic	8	24.2%
Dyspnea	3	9.1%
Exercise intolerance	6	18.2%
Sleeping disorder	1	3%
Chest pain	2	6.1%
More than one	13	39.4%
	N=33	%
Congenital anomalies		
No	22	66.7%
Double outlet right ventricle	2	6.1%
Bilateral superior vena cava	2	6.1%
Right sided aortic arch	5	15.2%
Patent ductus arteriosus	1	3%
Atrial septal defect	1	3%

Table (2): Distribution of right and left ventricle volumetric parameters, and Aortic artery parameters in the studied patients:

	N=33	%
Right ventricle systolic function		
Good	15	45.5%
Fair	7	21.2%
Impaired	11	33.3%
	Mean ± SD	**Range**
Ejection fraction (%)	50.88 ± 10.42	22 – 66%
EDV (ml)	221.91 ± 89.82	92 – 418
ESV (ml)	115.15 ± 66.15	33 – 276
SV (mL/m2)	106.73 ± 32.4	57 – 180
EDVI (mL/m2)	152.48 ± 41.12	89 – 232
ESVI (mL/m2)	72.46 ± 27.64	30 – 127
SVI (mL/m2)	79.3 ± 22.89	39 – 135
	N=33	%
Left ventricle systolic function		
Good	22	66.7%
Fair	5	15.2%
Impaired	6	18.2%
	Mean ± SD	**Range**
Ejection fraction (%)	56.24 ± 9.65	30 – 72%
EDV (ml)	119.3 ± 45.61	44 – 229
ESV (ml)	54.56 ± 29.81	12 – 144
SV	64.28 ± 19.86	22 – 112
EDVI	82.43 ± 19.58	46 – 126.5
ESVI	35.75 ± 12.12	16 – 66
SVI	46.79 ± 12.68	14 – 77
RT EDVI/LVEDVI	1.91 ± 0.63	0.86 – 4.17

Aortic artery parameters in the studied patients			
	Mean ± SD	Median (IQR)	Range
Aorta netflow (cm/sec)	59.11 ± 16.28		32 – 104
Aorta back (cm/sec)	1.61 ± 2.89	1.5(0 – 3)	0 – 13
Aorta forward (cm/sec)	60.71 ± 16.8		32 – 104
Aorta RF (%)	2.07 ± 4.03	0(0 – 4%)	0 – 17

EDV: End-diastolic volume, EDVI: End-diastolic volume index, ESV: End-diastolic volume, ESVI: End-diastolic volume index, SV: Stroke volume, SVI: stroke volume index, EDVI: end systolic volume index. RF: regurgitant fraction.

Table (3): Distribution of pulmonary arteries & tricuspid valves regurgitation status in the studied patients:

	N=33	%
PV regurgitation		
RF<20 (mild)	5	15.2%
RF 20 – 40% (moderate)	11	33.3%
RF>40% (severe)	17	51.5%
Tricuspid regurgitation:		
No	2	6.1%
Mild	18	54.5%
Moderate	10	30.3%
Severe	3	9.1%
	Mean ± SD	Range
RPA netflow	36.73 ± 12.84 ml	18 – 62
RPA back	12.86 ± 9.8 ml	0 – 37
RPA forward	49.5 ± 17.78 ml	18 – 85
RPA RF (%)	23.77 ± 15.55	0 – 59
LPA netflow	24.13 ± 10.38 ml	7 – 48
LPA back	11.3 ± 10.16 ml	0 – 46
LPA forward	35.43 ± 17.42 ml	16 – 95
LPA RF (%)	30.32 ± 17.46	0 – 59
MPA netflow	55.4 ± 19.89 ml	30 – 99
MPA back	36.43 ± 26.1 ml	3 – 125
MPA forward	91.83 ± 38.32 ml	53 – 194
MPA RF (%)	36.29 ± 13.61	5 – 52
RPA:LPA	1.8 ± 0.81	1.1 – 3.4

PV: Pulmonary valve, RF: regurgitant fraction, RPA: right pulmonary artery, LPA: left pulmonary artery, MPA: Main Pulmonary artery.

Table (4): Distribution of residual pulmonary arteries stenosis, interventricular septal flattening, and residual shunting in the studied patients:

	N=33	%
Residual pulmonary stenosis:		
No	6	18.2%
Mild	16	48.5%
Moderate	8	24.2%
Severe	3	9.1%
	Mean ± SD	Range
Pressure gradient	35.31 ± 16.19	16 – 81
	N=33	%
Septal flattening		
No septal flattening	11	33.3%
Septal flattening in systole	8	24.2%
Septal flattening in diastole	14	42.4%
Residual VSD		
Absent	23	69.7%
Present	10	30.3%
Qp:Qs ratio:		
No residual shunt (ratio 1)	20	60.6%
R-L shunt (ratio<1)	4	12.1%
Small shunt (ratio<1.5)	9	27.3%

VSD: ventricular septic defect, pulmonary (Qp) and systemic flow (Qs)

Table (5): Distribution of Late gadolinium enhancement (LGE) and grading protocol of right ventricle in the studied patients:

	N=33	%
LGE		
No enhancement	6	18.2%
RVOT enhancement	23	69.7%
VSD patch region	18	54.5%
RV insertion area	6	18.2%
RV surface of septum except for VSD patch region	5	15.2%
Inferior wall of RV	1	3%

RV: Right ventricle, LGE: Late gadolinium enhancement, RVOT: right ventricular outflow tract, VSD: Ventricular septic defect

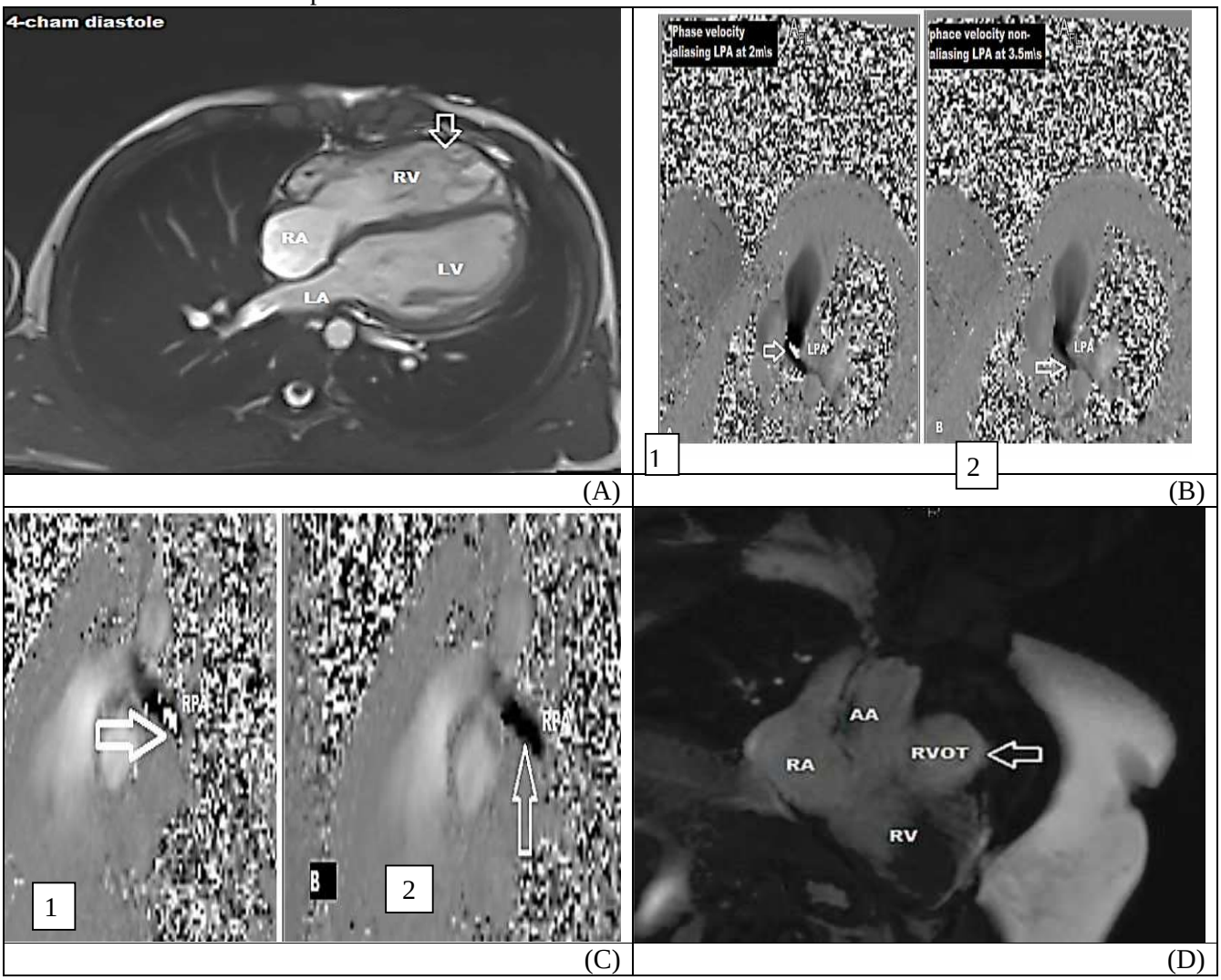

Figure 1: (A) MRI 4-chamber view during diastole shows RV dilatation (arrow), (B) MRI IP phase velocity study for LPA (B1) shows aliasing at 1.5m\s velocity (arrow), and (B2) shows non-aliasing at 3.5m\s velocity (arrow), denoting LPA stenosis, (C) MRI IP phase velocity study for RPA (C1) shows aliasing at 1.5m\s velocity (arrow), and (C2) shows non-aliasing at 3.5m\s velocity (arrow), denoting RPA stenosis, (D) MRI LGE sequences shows enhancement at RVOT likely site of patch (arrow).

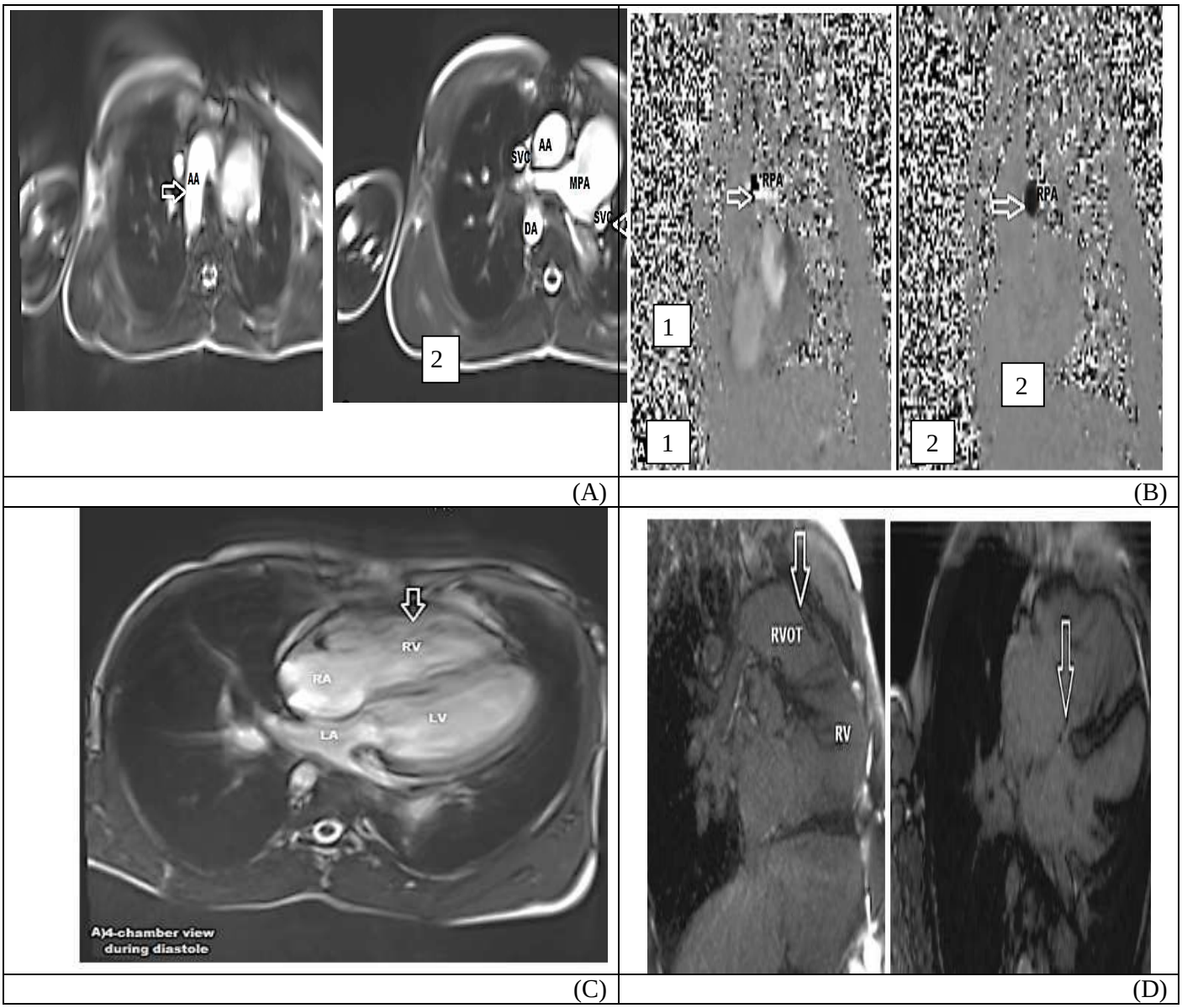

Figure 2: (A) MRI 4-chamber view during diastole show right ventricular dilatation (arrow), (B) MRI TP phase velocity study for RPA, (B 1) shows aliasing at 1.5m\s velocity, (B 2) shows non-aliasing at 3m\s velocity denoting RPA stenosis, (C) MRI axial view (C1) shows right-sided aortic-arch (arrow), (C2) shows persistence left SVC (arrow), (D) MRI LGE sequences, (D1) RVOT view shows enhancement at RVOT likely site of patch (arrow), (D2) 4-chamber view shows enhancement at the basal IVS (arrow).

DISCUSSION

The most prevalent type of congenital disability is congenital heart disease (CHD), which affects 1 percent of the population and almost always necessitates cardiovascular surgery within the first few months of life. About 1 in 3,500 newborns are born with tetralogy of Fallot (TOF), which is the most common cause of congenital heart disease in cyanotic children [9].

The RVOT obstruction seen in TOF has a clear embryological basis, with the antero-cephalad deviation of the developing outlet ventricular septum and the aberrant hypertrophied septo-parietal trabeculations that wrap the subpulmonary outflow tract being the most likely culprits.[10]. Sub-pulmonary stenosis, aortic override, a ventricular septal defect (VSD), and right ventricular hypertrophy are the four classic

features of this abnormality [11].

We analysed data from 33 patients admitted to the Radiology Department at Zagazig University Hospitals who had undergone repair for Fallot tetralogy. According to the socio-demographic data, our results showed that the age of patients ranged from 2 to 51 years, with a median age of 7.75 years. Eighteen patients were (<18 years); 16 cases (48.5%) were males, while 17 cases (51.5%) were females.

Fifty-six patients were included in the Attalla et al. [10] study; all of them had tetralogy of Fallot repaired surgically. Twenty of the fifty-six patients (35.7% of the total) had RV dysfunction, which aligns with our study's findings that more than half of the patients (18 out of 33 representing 54.5%) had RV systolic dysfunction. However, only three of the fifty-six patients (5.4%) were found to have LV dysfunction. Our sample size may be smaller than that of Attalla et al. [10], which could account for the discrepancy.

In agreement with our study, Similar to the findings of Attalla et al. [10], most cases (50/56, or 89.3%) were diagnosed with pulmonary regurgitation. Wherein the majority of patients (26/50, or 46.4%, of the 89.3%) had a severe degree of regurgitation, our study found that the majority of patients (17/33) were diagnosed with a severe degree of pulmonary regurgitation (representing 51.5 percent).

Three out of eleven patients who had correction of tetralogy of Fallot had regurgitation fractions of PV regurgitation of less than 40% (representing 27.2 percent), according to research by Saraya et al. [12]. Contrary to our findings, more than half of the patients had RF below 40%; This included 16 of 33 patients (48.5%). The difference between our study and Saraya et al. [12] can be attributed to the different sample sizes.

Most of our patients, 31 out of 33, had tricuspid regurgitation, which is consistent with the outcomes of Attalla et al. [10] as most patients, 49 out of 56, had tricuspid regurgitation (87.5%).

Kavurt et al. [13] reported pulmonary regurgitation in 95 of 110 symptomatic patients with corrected TOF (86.43 percent). However, in our study results, all patients (100%) developed pulmonary regurgitation with different degrees.

Most of our patients, 17 out of 33 (51.5%), had severe PA regurgitation, and less than half of the patients (45.5%) had good RV systolic function, with the mean EF (± SD) was 50.88 ± 10.42. This disagreed with a study involving a large group of patients with rTOF, which was done by Mercer-Rosa et al. [14], who found that the vast majority (85.0%) had mild pulmonary regurgitation assessed qualitatively by echocardiography and CMR, with a mean pulmonary regurgitation fraction by CMR of 34.2% and with a mean RVEF of 60.6% (8.2%), most people had mild to moderate PR with preserved RV shortening.

Eight out of thirty-three individuals in our study had aortic regurgitation (representing 24.2 percent). Similarly, Attalla et al. [10] found that only 11 of 56 patients had aortic regurgitation in fewer than a third of those cases (representing 19.6 percent).

In our study, less than one-third of the patients, 4 out of 33 (12.1%), had RV systolic dysfunction with EF less than 40%. This was not in line with the study of Saraya et al. [12], who showed that more than one-third of the patients had right ventricular failure in 4 out of 11 patients with EF less than 40% (representing 36.3% of total patients).

Our study found that residual VSD was present in fewer than a third of cases (10 of 33 individuals) (30.3%). 12.1% had an R-L shunt, and 27.3% had a small shunt. Eleven out of fifty-six patients exhibited a residual ventricular septal defect, consistent with Attalla et al.'s findings [10].

In our study, RVOT obstruction was evident in 27 of 33 patients (81.8 percent), manifested in various ways. Mild MPA stenosis was seen in 16 patients (59.3%), moderate MPA stenosis was present in 8 patients (29.6%), and severe MPA stenosis was present in 3 patients (11.1%), with a mean pressure gradient of 35.31±16.19 mm/Hg. This agreed with Attalla et al. [10]: 32 out of 56 had RVOT obstruction, and 24 patients were without RVOTO. Our results disagreed with Latus et al. [15], as 27 out of 54 patients studied showed RVOT blockage, while others did not. This was found in a study of patients

with corrected TOF (50 percent for each).

Our study results showed that the majority of the patients, 27 out of 33 patients, had RV LGE. Among the 27 patients, 23 (85.2%) had RVOT enhancement, 18 (66.7%) had VSD patch region LGE, 6 (22.2%) had RV insertion area enhancement, 5 (18.5%) had RV surface of septum except for VSD patch region LGE, and 1 (3.7%) had RV inferior wall enhancement. This was in agreement with Saengsin et al.'s [16] study, as later gadolinium enhancement was utilized, and all 127 patients with corrected TOF had RV LGE, the study found. One hundred and twenty-seven subjects (100%) had LGE in the basal superior segment, 82 (65%) in the middle superior segment, and at the mid anterior segment in 16 subjects (13%). While we were in disagreement with Ylitalo et al. [17], who used late gadolinium enhancement, All 40 patients with corrected TOF had RV LGE, and in the vast majority (39 of 40), RV LGE was also present outside of the surgically damaged areas. There was a favorable correlation between RV end-diastolic volume, pulmonary regurgitation velocity, and the degree of LGE. Post-operative follow-up time was also a factor in whether or not LGE was present.

Our study had some limitations, including that the duration of the study was short, so few cases of operated Fallot tetralogy were collected. A longer duration study with a bigger sample size would show a better picture. Chances were that only the complicated cases detected by Echocardiography were referred for MRI examination, which could also have introduced some bias. The lack of detailed clinical information for some cases (ECG characteristics, functional capacity, and echocardiogram) could contribute to the analysis of the information obtained by MRI. A complete assessment data would give better results. So, further studies are needed to compare CMR findings with other cardiac function measures to provide a more comprehensive assessment. Future studies should investigate the impact of postoperative complications on cardiac function and long-term outcomes.

CONCLUSION

Tetralogy of Fallot patients with surgical correction benefit greatly from CMR for follow-up evaluation. It aids in assessing surgical repair effectiveness, identifying residual problems, and directing subsequent operations by giving thorough anatomical and functional information. Its superior imaging capabilities and lack of invasiveness make it a valuable tool in the care of TOF patients.

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FIGURE LEGENDS

Figure 1: (A) MRI 4-chamber view during diastole shows RV dilatation (arrow), (B) MRI IP phase velocity study for LPA (B1) shows aliasing at 1.5m\s velocity (arrow), and (B2) shows non-aliasing at 3.5m\s velocity (arrow), denoting LPA stenosis, (C) MRI IP phase velocity study for RPA (C1) shows aliasing at 1.5m\s velocity (arrow), and (C2) shows non-aliasing at 3.5m\s velocity (arrow), denoting RPA stenosis, (D) MRI LGE sequences shows enhancement at RVOT likely site of patch (arrow).

Figure 2: (A) MRI 4-chamber view during diastole show right ventricular dilatation (arrow), (B) MRI TP phase velocity study for RPA, (B 1) shows aliasing at 1.5m\s velocity, (B 2) shows non-aliasing at 3m\s velocity denoting RPA stenosis (B) shows non-aliasing at 3.5m\s velocity (arrow), denoting LPA stenosis, (C) MRI axial view (C1) shows right-sided aortic-arch (arrow), (C2) shows persistence left SVC (arrow), (D) MRI LGE sequences, (D1) RVOT view shows enhancement at RVOT likely site of patch (arrow), (D2) 4-chamber view shows enhancement at the basal IVS (arrow).

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